Amebic Granuloma of Cecum

Report of an Intractable Case

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THERE ARE relatively few diseases that respond dramatically to a therapeutic agent. Some, however, possess such specificity of response that the result is utilized as a diagnostic test. For example, Snell[7] stated that for every diagnosis of amebiasis made by the finding of ameba in the stool, one is made by a therapeutic trial with emetine. He further remarked that "there is no disease condition which responds to specific treatment in a more dramatic manner." Silverman and Leslie,[6] in reference to amebic granuloma, added that the outstanding and almost universal characteristic of these lesions is their response to specific therapy. The "rapid resolution of these tumors," they said, "can only be compared with the melting away of syphilitic gummas after exhibition of the iodides." They noted, furthermore, that such granulomas disappear a week to ten days after administration of emetine. Spicknall and Peirce,[8] in commenting on a review of the literature on amebic granuloma, observed that the great majority of so-called "amebomas" disappeared completely within a month from the beginning of treatment.

The case of amebic granuloma reported herewith is presented for several reasons. First, it is at variance with the usual immediate salutary response to antiamebic agents repeatedly mentioned in the literature (complete resolution took several years); second, the unusual opportunity was afforded to observe the slow resolution of the lesion by serial roentgenographic study over a six-year period; and, third, it calls attention of the surgeons and clinicians to a lesion which must be given serious consideration in the differential diagnosis of cancer of the colon.

CASE REPORT

The patient, a 37-year-old veteran, was first admitted to the Veterans Administration Hospital, Oakland, October 3, 1952, because it was thought he might have a liver abscess. In July of 1945, while on military duty in the Philippines, the patient had had bloody diarrhea with fever, sweats, malaise and abdominal pain. He was treated with sulfaguanidine. Improvement took place but some diarrhea remained until the patient was discharged from the army in November 1945. A month later diarrhea became more severe and he was hospitalized at a military institution. At that time E. histolytica was found in the stool. He was given emetine, carbarsone and sulfasuxidine, but the record did not show the dosage. Dramatic improvement resulted. A year later diarrhea recurred. A private physician found E. histolytica in the stool and emetine was given. The condition improved. During the next six years, diarrhea recurred periodically and the patient received emetine each time, with improvement. So far as the patient knew, no other antiamebic drug was administered. Around Christmas, 1951, an episode of abdominal pain led to appendectomy. The postoperative course was uneventful. A week before the patient was admitted to the Veterans Administration Hospital in Oakland, loose bowel movements again returned, and fever, drenching night sweats, chills, cough and pain in the right upper quadrant of the abdomen soon followed.

Further data received from the Veterans Administration Regional Office revealed that in 1948 a barium enema showed a deformity of the cecum, which was irregular, constricted and presented ragged margins. The same conditions were noted in a roentgen study in 1950. Cysts of E. histolytica were found in the stool at that time. The patient's medical record contained note of episodes of rapid heart action and of two attacks of "nervous collapse."

Upon physical examination the patient was observed to be moderately undernourished, apprehensive, and chronically ill. The oral temperature was 100.6° F. and the pulse rate was 104. There was pronounced pain on compression of the right lower rib cage. Tenderness and guarding were noted in the right upper quadrant of the abdomen along the costal margin. The edge of the liver was not felt.

In an x-ray film of the chest, increased markings were noted in the right lower lobe. Fluoroscopically observed, the right leaf of the diaphragm moved only one-third of normal excursion. It was not elevated. A barium enema on October 8, 1952, showed considerable spasm with poor haustration. The cecum was deformed, irregular and greatly narrowed. The roentgenologist's impression was that the patient had diffuse amebic colitis with probable granuloma of the cecum. A barium enema on October 29, 1952, after antiamebic therapy, showed less deformity of the cecum.

Leukocytes numbered 19,500 per cu. mm. of blood, with 17 band forms. The hemoglobin content was 14.5 gm. per 100 cc. Serum bilirubin was 1 mg. per 100 cc. There was 12.5 per cent retention of bromsulfalein in 45 minutes. The result of a cephalin flocculation test was negative. E. histolytica was present in the stools in great number. Subsequent stools after treatment were all negative for parasites.

A diagnosis of amebic colitis with probable liver abscess was made and antiamebic therapy was started. This consisted of chloroquine, 1 gm. daily for two days followed by 0.5 gm. daily for 19 days in two divided doses each day; diodoquin, 3 capsules three times a day (a total of 1.89 gm. daily) for three weeks, and aureomycin, 250 mg. four times a day for ten days. At the completion of this simultaneous therapy, ten daily injections (0.065 gm. each) of emetine were given. Five days after institution of treatment the patient was afebrile and considerably improved. He was discharged seven weeks after entry. A follow-up barium enema study on January 9, 1953, showed further improvement of the conditions

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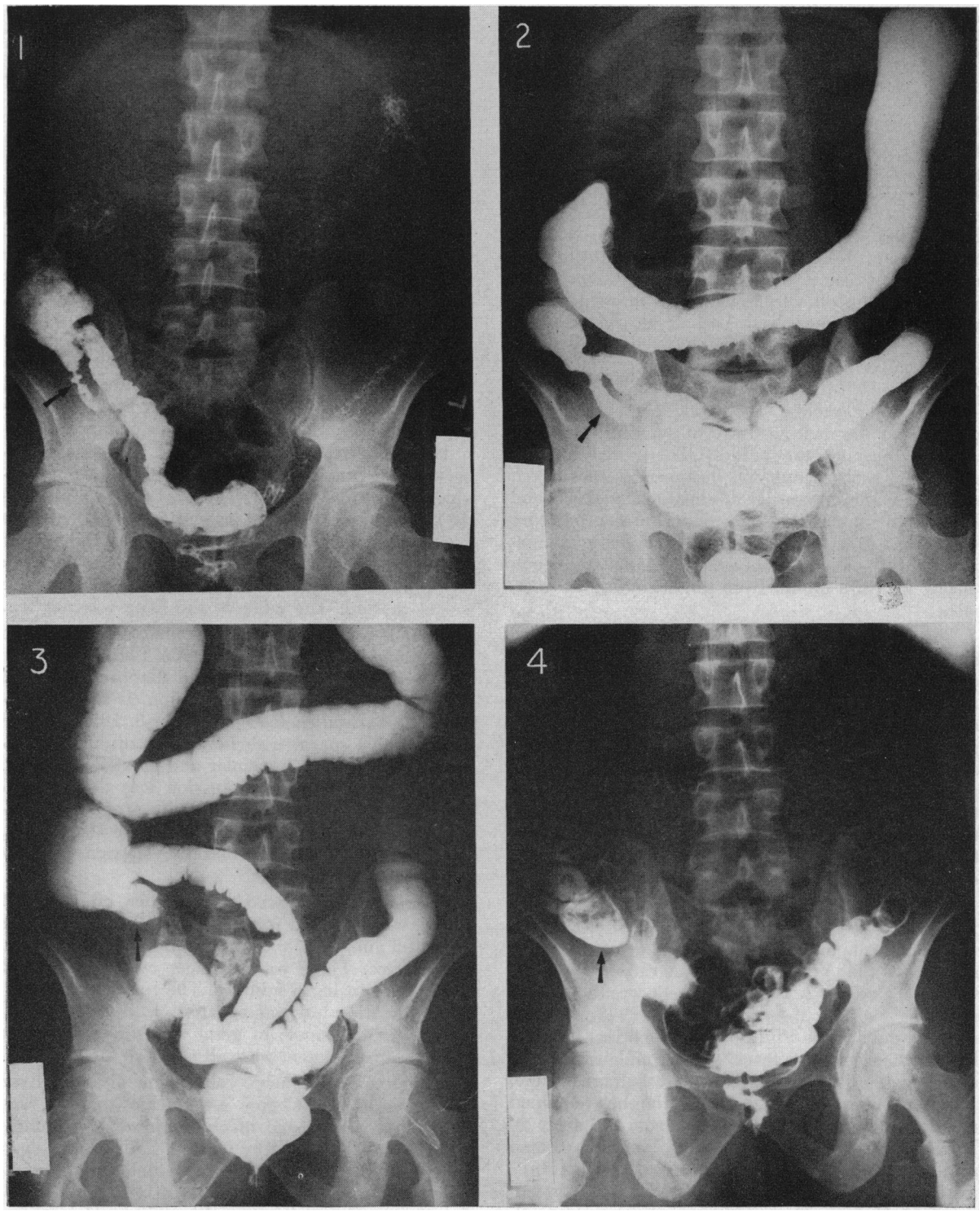

(1) Initial roentgenogram in 1948, showing deformity of cecum with narrowing and irregularity. (2) (1952) Study, after intermittent emetine therapy (only). Cecum deformed, narrowed and "conical." Ileocecal valve abnormally patent. Distal colon shows poor haustrations, and fine irregularity suggestive of ulcerations. Interpretation by radiologist: Diffuse amebic colitis and granuloma of cecum. (3) (1953) Improvement, with better filling of cecum, after several courses of adequate therapy. Cecal deformity still present, as is involvement of mucosa of proximal half of colon. (4) (April, 1954) Cecum now fills out normally on air contrast study. No constant defect. Findings essentially normal with resolution of previous granulomatous changes.

noted in the colon, but still considerable deformity of the cecum.

Second admission. The patient reentered March 19, 1953, because of weakness, fatigability and abdominal pain. He did not appear ill on this occasion. There was guarding of the abdomen. The edge of the liver was barely palpable. In the right lower quadrant of the abdomen a moderately tender, slightly irregular and immobile mass was felt.

Chest x-ray films, blood cell counts, sedimentation rate and liver function tests were within normal limits. Several stools were negative for ameba. A barium enema examination showed conditions similar to those of the previous study, again with deformity of the cecum. The patient refused to have sigmoidoscopic examination. Because of the abdominal mass and the persistent barium enema findings, a presumptive diagnosis of amebic granuloma of the cecum was made. The patient was thereupon given another course of emetine and diodoquin, and then a three-week course of chloroquine (same dosage as on first admission). He was discharged April 1, 1953. Another barium enema on May 15, 1953, showed additional improvement.

Third admission. The patient was readmitted July 21, 1953, for follow-up examination. At this time there were minimal changes from before. The abdominal mass had regressed but was still palpable. Another three-week course of chloroquine and diodoquin was given (dosage as before).

Final admission. Admitted again April 27, 1954, the patient said he had a feeling of being "run down," had had dizzy spells and a recent "blackout." In addition, he complained of diarrhea and abdominal pain. The temperature was 98.4° F. The mass previously noted in the right lower quadrant of the abdomen could no longer be palpated. The stools were negative for ameba. A barium enema showed nearly complete resolution of the changes in the right colon. An air contrast study disclosed that the cecum distended normally and there was no constant defect of outline. The conclusion by the roentgenologist was that "findings are now essentially normal, with resolution of the previous granulomatous changes."

It was concluded that the probable amebic granuloma had finally resolved after repeated courses of therapy over a period of several years.

DISCUSSION

Anderson, Bostick and Johnstone,[1] in their text on amebiasis, said that when an ameboma has been in existence for a long time and is secondarily infected with bacteria, fibrosis and distortion of the colon may delay or prevent the disappearance of the mass after antiamebic therapy. In some cases, excision may be necessary. They expressed belief that ameboma is due to repeated amebic invasion plus superimposed bacterial infection. Because of the latter, it has been advised that a broad spectrum antibiotic be added to the usual therapeutic regimen. Other observers have also noted delayed response of such granulomas to therapy.[9]

Amebic granuloma occurs most commonly in the cecum. It cannot be distinguished radiologically from cancer, giving rise to an important problem, as attested by a number of postoperative deaths in the presence of unsuspected amebic granuloma.[2,4,5,6,8] A course of antiamebic therapy preoperatively may be life-saving in such cases and should be administered in any case in which the possibility exists. The danger of surgical treatment in amebic disease was first brought out during the disastrous 1933 Chicago epidemic when the mortality rate for intestinal operations in the presence of amebiasis was 40 per cent. Many of the patients were operated upon for acute appendicitis, appendiceal abscess or neoplasm of the colon.

Amebiasis in the United States usually is a chronic, low-grade, dormant disease, often without diarrhea. For the latter reason many cases are attributed to functional disturbance and stools are not examined. In some such cases the proper diagnosis is reached when radiologic examination of the cecum is carried out. Wilbur and Camp[9] found 21 suspicious cases in a review of 300 barium enema examinations. In 12 of the 21 cases, E. histolytica was found in the stools. It was the opinion of these investigators that roentgenologic study of the colon is the most valuable method of finding amebic disease of the colon. The significant roentgenologic changes in the cecum as described by these observers consisted of spasm, dilatation, relaxation, and abnormal patency of the ileocecal valve; inflammatory induration with "coning" and inflammatory tumefactive defects. The earlier changes involved the mucosa, the sharp, smooth character of the cecal walls being replaced by a finely granular, irregular contour of the mucosa. There was often an associated cecal spasm. Wilbur and Camp noted carefully that these changes are not pathognomonic since they may occur also in other inflammatory diseases. Golden and Ducharme[3] stated that recognizable deformity of the cecum is likely to be present in over one-third of the cases of amebiasis. They noted that in many cases the initial suspicion of this disease came from the roentgenologist.

SUMMARY

A case of amebic granuloma (ameboma) of the cecum requiring six years for resolution is presented. Roentgenologic studies were of interest, for slow and progressive improvement was shown.

The usual dramatic and rapid response of amebic granuloma to specific therapy may not occur, especially in cases not adequately treated at the outset and in which extensive fibrosis and secondary infection occurs.

Amebic granuloma may simulate neoplasm of the colon. Where suspected, a therapeutic trial with antiamebic agents is indicated before operation, since a high mortality rate is associated with surgical intervention in the presence of amebiasis.

Amebiasis may be suspected on the basis of roentgenologic findings in the cecum. "Coning" is said to be the most suggestive sign.

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Congenital Bladder Neck Contracture In Male Siblings

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ONLY PASSING REFERENCE to bladder neck contracture in siblings is found in the literature. Campbell[6] mentioned this disorder in twin brothers, but did not give details. The present report describes the salient findings recently observed in two young brothers who were uremic from advanced renal damage due to bladder neck contracture and discusses the improvement in their clinical condition following open surgical correction of the vesical obstruction.

FUNDAMENTAL CONSIDERATIONS

Evaluation of distal urologic conditions in children involves consideration of *congenital, acquired, extrinsic* and *intrinsic* lesions.[1] Obstructive mechanisms noted by Campbell were *urethral meatal stenosis, congenital contracture of the vesical outlet, congenital valvular obstruction of the prostatic urethra,* deep urethral urinary blockage by *polyp, ureterocele, trigonal curtain, prostatic enlargement* and, of high incidence, *neuromuscular vesical disease.*[6,7] Burns agreed essentially with that listing and with that order of frequency.[4] However, he minimized the clinical incidence of urethral valves, noting that these occurred in only three of eighty-one patients reviewed by him and his colleagues.[4]

Bladder neck contracture is three to five times more common in boys than in girls. Other congenital anomalies, either in the genitourinary tract or elsewhere, may coexist with any one or several of these obstructive lesions; most common of these concomitant distal urologic conditions are urethral meatal stenosis and bladder neck contracture.

In 1915 Beer suggested that fibrosis of the neck of the bladder of a young patient may be a secondary change in the spastic sphincter which results from a fundamental neuromuscular disease.[3] Campbell reported verification of Beer's postulate and demonstrated histopathologically that bladder neck contracture develops from submucous fibrosis and smooth muscle hypertrophy, often involved by round cell infiltration.[6]

Symptoms of bladder neck contracture may occur early in life (since excretion of urine starts in the fifth and sixth month *in utero*) or they may be delayed until even the third decade. Urinary frequency, straining to void, dribbling and/or enuresis are prominent manifestations. Protracted, unsatisfactorily explained gastrointestinal symptoms may indicate urinary tract disease. The signs are those of recurrent urinary infection associated with residual urine. Degree of obstruction and upper urinary tract damage are not directly reflected by the quantity of residual urine, which may vary from several ounces to several liters. Only rarely is acute urinary retention the first symptom of an obstructive urological condition. Eventually, systemic features of progressive renal failure, such as anemia, malaise, anorexia and failure to gain weight become predominant clinical signs.

Diagnosis is established by history, by physical examination (vesical or renal "abdominal masses") and by full urological study. Urinalysis, measurement of residual urine, bacteriological culture (with antibiotic sensitivity tests) and phenolsulfonphthalein excretion tests are fundamental procedures to be carried out. Blood tests to appraise uremia and electrolyte balance are required. Intravenous urography will demonstrate the degree of renal, ureteral and vesical damage but it usually affords only inexact information as to location and type of distal urinary disease. Cystography is probably the most valuable radiographic procedure; a voiding cystogram may demonstrate ureteral reflux that might be missed by retrograde cystography alone. Cystourethrography is a refinement advocated by some investigators.[4] Cystoscopy, necessarily panendoscopy, is most helpful and will usually demonstrate trigonal hypertrophy. The presence of a *bas-fond* is of value in delineating chronic infra-vesical obstruction as etiological.[6] Trabeculations are usually absent in

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